AMENDMENTS TO THE SPECIFICATION

Please replace the three paragraphs at page 5, lines 6-25, with the following amended paragraphs.

~~Fig. 1A~~. Fig. 2A. Sepharose beads bound with purified glutathione S-transferase (GST, lane 2) and GST fusions of Hec1 containing amino acids 56-642 (GST-hsHeclp, lane 3) or 251-618 (GST-15Pst, lane 4) were mixed with in vitro translated, ^{35}S-methionine-labeled Nek2 (lane 1), then washed extensively. The binding complexes were separated by SDS-PAGE, dried, and visualized by autoradiography.

~~Fig. 1B~~. Fig. 2B. Specific regions of Hec1 bind to Nek2 by yeast two-hybrid assay. Deletion mutants containing the different coiled-coil domains of Hecl were fused in-frame to a GAL4 DNA binding domain. Nek2 was expressed as a GAL4 transactivation domain fusion. Yeast transformants with these two hybrid proteins were grown in liquid cultures and used for O-nitrophenyl-β-galactopyranosidase quantitation of β-galactosidase activity. The fold-increase in activity compared to the host yeast strain Y153 is indicated. Assays were done in triplicate for each transformation.

~~Fig. 1C~~. Fig. 2C. Cell cycle-dependent interaction between Hec1 and Nek2. T24 bladder carcinoma cells were first density arrested at Gl (lanes 2) and then released for re-entry into the cell cycle. At different time points after release from density arrest (indicated above the lanes), cells were collected and lysed. The clarified lysates were immunoprecipitated with mAb9G3 anti-Hec1 monoclonal antibodies (upper two panels) or anti-Nek2 antisera (lower two panels). Hec1 and Nek2 co-immunoprecipitated at G2 and M phases (lanes 5 and 6).